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THE
LARYNGOSCOPE.

VOL. LXIII APRIL, 1953 No. 4

GLOMUS JUGULARE TUMOR OF THE MIDDLE EAR. CLINICAL ASPECTS.*

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Prior to 1945, the tumor now known as Glomus Jugulare or Tympanic Gland Tumor was known as such to Guild.[1] In 1945, Rosenwasser[2] wrote about it. Within the next five years, it ripened into a leading topic in otolaryngology, it developed considerable interest in the field of pathology, and it even stimulated a little curiosity among neurosurgeons. Now, in the almost eight years that have followed Rosenwasser's (might I say) discovery, there has been a snowballing effect on the literature as far as this tumor is concerned.

Histologically, this tumor has been studied extensively by many pathologists, including Otani[3] and Lattes.[4] Physiologically, there has been much talk about the carotid body, and similar tissue, but the answer is still hazy. My purpose in bringing up this subject again is to elaborate on the clinical aspects as I have observed them in six patients, whose diagnoses are proved by biopsies, and who have been or are being treated by surgical or X-ray therapy, and in some instances by both methods. Five other patients have met the clinical requirements for diagnosis, but are not proven cases of glomus jugulare tumor, because there has not been histological examination of the questionable tissue.

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Nashville, Tenn., Jan. 12, 1953.

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Of the six "biopsy positive" patients, there were four males and two females. The ages varied from 26 to 50 years, with an average of 36. Strangely, the right ear was involved every time. Of the five "clinically positive," but not histologically proven, patients there were four females and one male. The ages varied from 27 to 72 years, with an average of 44. The left ear was involved four times, and the right ear once. This writing will be concerned only with those six patients who are "biopsy positive."

The interval from the onset of the first symptom to the time that I first saw each one of these patients was three years, 20 years, seven years, 14 years, six months and five years.

The pathologists who have borne the burden of making the diagnoses in these cases are Dr. Ivan Ross, of the Georgia Baptist Hospital, Dr. Abner Golden, of the Emory University Hospital, and Dr. Merrill Whorton, of the Veterans Administration Hospital, all in Atlanta, Ga. Collaborating radiologists are Dr. W. W. Bryan and Dr. Leonard Long, both of Atlanta, Ga.

We realize that there is nothing new about the tumor, not only because the Bible states that "there is no new thing under the sun" (Ecc. 1:9), but also because, prior to 1945, these tumors were recognized, although they were classified under other diagnoses. Now, many have been re-evaluated to be glomus jugulare. With the advent of concerted study in one direction, there have been brought together certain symptoms and findings, referable to this disease, which makes it possible to prognosticate a diagnosis, almost with certainty, before obtaining the biopsy report. In addition to these symptoms and findings, I should like to mention something of the surgical and postsurgical observations.

THE SYMPTOMS.

In this group, there were eight principal complaints, one or more of which caused each patient to seek medical advice. These will be discussed in the reverse order to the number of times that the complaint was mentioned.

1. Itching in the ear. One patient. A Negro woman, aged 50, stated that itching deep in the ear canal came on as the first symptom, or at the same time as the "swishing" noise. The itching caused her more "misery" than any other symptom.

2. Discharge from the ear. Three patients. This was purulent, thick and malodorous. It was the type of discharge that usually accompanies the chronically diseased ear. It aroused the suspicion of associated cholesteatoma.

3. Headache. Three patients. Headache was considered relevant only if the episodes were frequent, similar and unilateral to the side of the affected ear. Of these three patients, the headache was almost incapacitating in one and moderately severe in two.

4. Hemorrhage. Three patients. In two patients, hemorrhage occurred only when the ears were cleaned. One had used a bobby pin for the cleaning, and had procured what must have been a frightening blood-letting, since, according to the patient, "the blood squirted across the room." After careful questioning, it seemed that some blood did "squirt" on the patient's shoulder. One patient had spontaneous hemorrhage which occurred for the first time when he leaned over to pick up an electric motor.

5. Vertigo. Three patients. These patients stated that the vertigo, which consisted of fleeting attacks of being "lightheaded," was secondary in importance to other complaints.

6. Facial paralysis. Four patients. Four of these six patients complained of some degree of facial nerve involvement on the side of the affected ear. The degree ranged from slight twitchings or minor paralyses of some of the facial muscles to total, flaccid, homolateral paralysis.

7. Impaired hearing. Six patients. The complaints varied from scarcely noted impairment to total deafness in the affected ears.

8. Tinnitus. Six patients. This occurred in all cases, and in four instances it was the first symptom. Most inconsistent

was the type of tinnitus. One stated that her first ear noise sounded like a clap of thunder. Another said that she awoke in the middle of the night and thought someone was chopping wood in her back yard. Another, whose home is located close to a railway station, said that he awoke soon after going to bed, thought that he heard a train pulling out of the station, discoverered that it was noise in his ear when his wife assured him that there was no train. Each of two patients stated that the noise was "a swooshing" and that it could be "cut off" by pressing on that side of the neck. In addition, there were the usual "steam" tinnitus, "ticking in the head" and "pumping noises." For those patients whose disease was of short duration, tinnitus was very consequential, but for those whose disease was of long duration, other symptoms overshadowed, even though they did not obliterate the tinnitus.

THE FINDINGS ON CLINICAL EXAMINATION.

1. Mass in ear. In every case there was a mass in the ear, although it did not always protrude from the region of the eardrum. It may have appeared to be incorporated in the eardrum, lending a mulberry-like roughness with a deep red or deep blue color; or it may have assumed the form of a flat, red polyp lying against the external surface of the eardrum, in which case, on closer inspection, it could be seen to involve the membrane at some place, usually in its posterior portion. On the other hand, it may have presented as a protruding mass, sometimes extending to the lateral limits of the external auditory canal.

When the mass looks like a common aural polyp, at which time it is usually accompanied by purulent secretion, there may be confusion as to the type of disease present, which can lead to an embarrassing situation during the course of making a diagnosis. According to the literature and personal conversation with doctors and patients, there have been occasions when attempts to remove the "polyp" have been followed by hemorrhage so vigorous as to become a matter of excitement to the patient, and the cause of some concern to

the physician. This, then, brings up the question of how to differentiate clinically the glomus "polyp" from the usual aural polyp.

2. The pulsation sign. Spontaneous pulsation of the tumor, the auricle, or the region of the neck just below the ear, sometimes is present, but it is not a constant finding. It may be that the incidence of spontaneous pulsation occurs in direct proportion to the extent of the invasion of the tumor, whether in soft tissue or bone, but it is not in proportion to the size of that part of the tumor which is visible. In other words, the tumor which protrudes from the external auditory canal may not pulsate, and yet there may be spontaneous pulsation of the tumor which scarcely protrudes from the region of the middle ear. Under certain conditions, however, it seems that all of these tumors will pulsate. Of this group of six patients, spontaneous pulsation was present in two, but pulsation, under the proper conditions, could be elicited in every case, with one exception. (This exception was Case 1 in this series and, at that time, this sign had not been recognized. Consequently, no attempt was made to find it.) This involves what I have thought of as "the pulsation sign." By the use of a clinical test, pulsation can be elicited where there is no spontaneous pulsation, and spontaneous pulsation can be increased.

This test is carried out with the aid of the Bruening magnifying otoscope. The patient is placed in the sitting position. By fitting the external auditory canal with an aural speculum large enough to seal off the canal, and, while looking through the magnifying otoscope with a satisfactory light, if the air pressure is increased in the external auditory canal, through the otoscope, by means of compressing the attached rubber bulb, the tumor will be seen to pulsate, sometimes almost violently. As the air pressure is increased by more compression of the rubber bulb, the tumor will blanch, and the pulsation will decrease. Then, as the air pressure is reduced by less compression of the rubber bulb, the pulsation of the tumor will return, until the normal room pressure has been reached, at which time the pulsation will again subside, or return to

the pretest spontaneous pulsation; furthermore, if, while pulsation is being produced by this air pressure method, the carotid artery system of the neck, on the same side as the affected ear, should be compressed, blanching of the tumor and subsidence of pulsation will occur. There will be a return to pulsation of the mass when the carotid pressure is released. I have not found this sign to be present where the tumor is ordinary polyp or granulation. Where there is pulsation of the eardrum in acute otitis media, the pulsation will disappear, rather than become accentuated, when the air pressure is slightly increased. The only other situation under which I have observed "the pulsation sign" is that which occurs when there is a large area of exposed dura mater in the radical mastoid bowl.

The number of patients upon whom this test has been confirmed is small; nevertheless, when this sign is present, I believe that it can be stated with certainty that caution is indicated if manipulaton of the mass is contemplated.

3. Facial paralysis. Unilateral facial muscle weakness, on the side of the affected ear, was noted in each of three patients at the initial examination. Another patient developed it during nine months of preoperative observation. It was not present in two of the six patients.

4. Hearing impairment. Hearing impairment was observed in all six patients. On audiometric examination, it was found to vary from slight to total deafness in the affected ear. The amount of deafness was in proportion to the size of the tumor, and to the pathologic changes in the temporal bone as observed on X-ray examination.

5. Labyrinthine function. For this, the caloric (water) test was used. Obviously, technical difficulties were encountered when the tumor mass filled the external auditory canal. In those cases, the caloric test could be carried out, only after the tumor mass had been removed; therefore, the test was not helpful. It might be stated, however, that the vestibular function was present or absent as the acoustic function was present or absent.

6. X-ray examination. The X-ray examination of the temporal bones may or may not add to the diagnosis. In the earliest stages of the disease, no changes are to be seen in the films. As the disease progresses, the mastoid cellular structure becomes Roentgenographically cloudy, closely resembling the appearance of acute mastoiditis before bony destruction. The clouding is also present in the cells of the petrous bone, if such cells are present. By the time that there is total loss of function of the VIIth and VIIIth cranial nerves, on the side of the affected ear, the generalized clouding of the mastoid and petrous cells gives way to an almost complete demineralization, which is more easily seen on the anterior-posterior (Towne's) position. There is the appearance of dissolution, not spotty necrosis. Only the hard, membranous bone of the labyrinth may appear to be intact.

If there is any characteristic X-ray evidence of the glomus jugulare tumor, it is this picture of the mastoid and petrous cells being "rubbed into coalescence." There is not so much demineralization as is seen in localized areas in acute surgical infection of the mastoid cells; there is not the sclerosis that appears in long-standing chronic infection of the mastoid bone; there is not the complete obliteration of bone as is seen in cancer of the temporal bone.

The question has arisen as to why there were changes in the mastoid cells, as noted at surgery and on X-ray examination, when by microscopic examination, no elements of the glomus tumor were found in the bone or the soft tissue of the mastoid process, posterior to the facial ridge.

Jones and Roberts[5] have pointed out that "if the blood to a bone is decreased, the bone undergoes increased calcification; if the blood supply is increased, the bone undergoes decalcification." On this basis, it is conjectured that the high vascularity of this tumor might cause enough hyperemia in the temporal bone to result in decalcification of its bony cellular structure. Then, the X-ray evidence of dissolution of the temporal bone might not indicate invasion by tumor, but rather an effect indirectly caused by the tumor. If this should be

true, the otologist would feel more the necessity of eradicating the disease, if possible, even in the late stages, with the hope of eliminating the focal point of hyperemia, and by this means, to stop the dissolving process in the bone. The pathologic nerve changes, unless very recent, probably would be irreversible.

7. Biopsy. This is necessary for confirmative evidence; therefore, the otologist should secure a good specimen of tissue. If the tumor is glomus jugulare, the bleeding at the time of procuring the tissue for biopsy will be severe, but it can be controlled by packing the external auditory canal with Gelfoam and plain gauze.

8. Arteriogram. Three patients. A study of the carotid artery system on the side of the affected ear, by injecting an X-ray opaque solution into the common carotid artery, was done in three cases. The concomitant X-ray studies did not demonstrate any abnormal condition.

TREATMENT.

There are three plans of attack on this tumor: surgical removal, X-ray therapy, or a combination of these two.

The question of injecting a sclerosing solution into the tumor was considered. This was done in two cases. Etalate (Parke, Davis and Co.) was used; 0.2 cc. of the 5 per cent solution was injected into each tumor mass, in the external auditory canal, on each of three occasions. No changes were noted in the tumors.

THE PROCEDURE OF OPERATION.

The endaural and the postauricular approaches have been used in the five operated cases. There does not seem to be any particular advantage to the use of the endaural approach, except for the elimination of the added amount of work required in making and suturing the larger postauricular incision. There are advantages to the use of the postauricular incision. There is wider exposure. Initial hemorrhage from the incident touching of a large, protruding glomus tumor is

obviated. It has been my experience that there is to be found a large blood vessel, in the posterior wall of the membranous external auditory canal, which runs horizontally from the region of the eardrum, laterally. This vessel can cause considerable "nuisance bleeding." It may be encountered when the posterior portion of the external auditory canal is severed from the pinna.

From the standpoint of the surgical treatment of glomus jugulare, the posterior two-thirds of the mastoid cell region bleeds no more than it does in infection of the mastoid process. For this reason, the radical mastoid cavity is prepared first. The middle ear is the location of the origin of the glomus mass, and surgical manipulation there is accompanied by profuse bleeding. As the operation on the tumor proceeds, there must be continuous and efficient aspiration of blood in order to obtain even fair visibility.

In these surgically treated cases, each tumor has been lifted from the periphery of the middle ear by using a narrow, blunt elevator. When freed from the periphery, the tumor mass has been separated from the middle ear region by the use of the elevator, or it has been lifted out of the middle ear with the aid of small grasping forceps. Following the removal of the tumor, the bleeding has been controlled with greater ease than expected, by packing the middle ear with a small amount of Gelfoam and plain gauze. A transfusion of 500 cc. of whole blood has been given during the course of each operation.

The radical mastoid bowl and the postoperative care have been handled in the routine manner. X-ray therapy has been started seven to 10 days following the operation. The dosage has varied from 2,500 r. to 4,800 r., according to the Roentgenologist in charge.

COMMENT.

In none of these five operated cases was there an obvious defect in the bone of the tympanum. It is doubted that in any instance was the dome of the jugular bulb removed. In two of the cases the tumors seemed to be cleanly enucleated, while

in the other three the tumors were removed piecemeal. Immediate postoperative hemorrhage was more easily controlled in the former.

In each of the three cases where the tumors were removed piecemeal, there was tumor invasion of the bone around the descending portion of the facial nerve, the tumor apparently invading from the posterior aspect of the middle ear at the level of the stapes; furthermore, in each of two of these cases, glomus tissue was removed from the descending portion of the facial nerve (or its sheath). Both patients had partial paralysis of the facial muscles on the side of the affected ear, prior to any manipulation. In each operation, the nerve in its sheath was surgically exposed above and below the point at which tumor tissue could be seen in conjunction with the nerve, and the tumor tissue was removed to the level of the nerve, without disturbing the nerve. In neither case was there any postoperative change in the partial facial paralysis. In one of these two cases, the bone around the descending portion of the nerve gave rise to profuse hemorrhage, although it was not so active as that in the middle ear. The microscopic examination of the soft tissue, from the middle ear and from the bone immediately posterior to the nerve, demonstrated malignant paraganglioma which had invaded bone.

Ligation of the external carotid artery may not serve to control the hemorrhage which is encountered when the tumor is being removed from the middle ear. Ligation of the external carotid artery, distal to the superior thyroid artery, but proximal to the ascending pharyngeal artery on the side of the affected ear, was performed on one patient immediately before the glomus operation was carried out. Bleeding seemed to be as severe as it was in each case where the artery was not ligated.

Approximately one month after operation, the "pulsation sign" could be elicited in the posterior inferior quadrant of

the affected middle ear in one patient, but it was not present in the other four. The significance of this finding, if any, is not known to me at this time.

All six "biopsy positive" glomus jugulare patients have been, or are being, treated. Five of these patients have been treated by surgical removal of the tumor, and three of these five have received X-ray therapy. The sixth patient is now completing a course of X-ray therapy, and there has not been any surgical procedure except for the removal of tissue for biopsy. The length of time since the beginning of treatment upon these patients varies from three months to four and one-third years, which is so short a period that no definite opinion can be formed regarding a so-called recurrence, which probably would more correctly be called a continuation of growth.

SUMMARY.

1. The symptoms and clinical observations of six patients who had or have the condition known as *glomus jugulare tumor of the middle ear,* as proven by biopsies, are presented.

2. Five other patients presented similar symptoms and findings *clinically* diagnostic of this condition, but biopsy report was not available on any of them.

3. The most constant, first noticed symptoms were *tinnitus* and *impaired hearing.*

4. The most constant clinically diagnostic finding was the so-called "pulsation sign."

5. Surgical enucleation of the tumor mass, together with X-ray therapy to the region of the affected middle ear, constituted the treatment.

6. It should be repeatedly emphasized that no estimate of the value of the therapy is as yet worthwhile. The answer will require observations over a period of many years.

GLOMUS JUGULARE PATIENTS.

‡*1.* R. B. H., 30-year-old man, first seen Sept. 9, 1948.
2. C. T., 27-year-old woman, first seen Aug. 8, 1949.
**3.* J. G. J., 33-year-old man, first seen May 23, 1950.
4. E. L. B., 39-year-old woman, first seen Sept. 20, 1950.
**5.* J. S., 38-year-old man, first seen Nov. 16, 1950.
**6.* W. M. G., 26-year-old man, first seen Aug. 7, 1951.
7. J. H. H., 35-year-old woman, first seen Nov. 27, 1951.
8. B. A. C., 72-year-old woman, first seen May 26, 1952.
**9.* R. E. C., 41-year-old woman, first seen Sept. 29, 1952.
†*10.* V. M., 50-year-old colored woman, first seen Nov. 4, 1952.
11. C. L. H., 46-year-old man, first seen Jan. 16, 1953.

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137 Doctors Building.

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*Patients biopsied.

†One patient, biopsy positive, seen in consultation with Dr. Nathan Gershon, of Atlanta, Ga.

‡This patient's biopsy was first read as squamous carcinoma; this was disputed by a second pathologist; the slides were sent to the Army Institute of Pathology, where a diagnosis of glomus jugulare was made.

THE RELATION OF SEPTUM RECONSTRUCTION TO EXTERNAL DEFORMITIES OF THE NOSE.*

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Abnormal nasal physiology due to recent or old injuries is the problem of the rhinologist. Current thought demands that the nasal function be restored to normal as nearly as possible; moreover, it is becoming more widely recognized that correcting a deformed or injured nose is a physiologic problem, not merely a cosmetic one.

Even today it is not unusual to treat severe nasal injuries associated with external deformities by realignment of the nasal pyramid with postponement of correction of the septum until a later date. On the other hand, in the case of a patient with a deformity due to an old injury, the rhinologist may be prone to repair the septum and refer the patient to another specialist for realignment of the pyramid. Admittedly, in some of these cases it may be better to repair the condition in two stages, but these can and should be done by the same surgeon. Surgical reconstruction of the septum may be performed along with surgical correction of such associated structures as the upper lateral cartilages, bony pyramid and lobule, which contains the lobular or lower lateral cartilages, columella and alae. Articles by Cottle,[1-4] Salinger,[5] Maliniac,[6] Fomon *et al.*[7] and the author[8-9] have emphasized the importance of this combined surgery.

Only enough of the anatomy will be discussed here to bring to your attention once again the importance of the upper lateral cartilages and lobule. The upper lateral cartilages are attached to the posterior surface of the nasal bones. They are in reality wings of the septal cartilage, of which they are an

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integral part, and together with which they form the cartilaginous vault. The relation of these cartilages to the septum varies from above downward. In its upper portion the cartilage is thicker, possibly for support, and as it approaches a lower level it becomes more pliable and quite frequently it is not joined to the septum at its most caudal position. This anatomic arrangement enables the lower part of the cartilage to move in and out from the septum in a valve-like manner.

The lobule contains the lobular (alar) cartilages, the columella, the alae and the vestibule. Actually, the lobule is an organ by itself, connected to the other parts of the nose only by skin externally, connective tissue fibres and skin inside; it plays an important part in normal nasal function. The lobular cartilage is somewhat U-shaped; its medial and lateral crura are connected by a dome, which, together with its mate of the other side, forms what is generally called the tip. The medial crura help to form the columella, and the lateral crura help support their respective alae.

The cephalic margin of the lobular cartilage overlies the caudal end of the upper lateral cartilage. The action of these two cartilages plays an important part in normal breathing. The upper lateral cartilages in their relationship to the septum are affected greatly by the lobular cartilages and the rest of the lobule. The size, shape and position of the nostrils are also important in this particular relationship.

The inner part of the lobule is known as the vestibule. The columella and membranous septum separate the lobule into two vestibules, and it is through these regions that all air must pass before it can enter the nose proper. Within the vestibule are several prominences which have the action of delaying the rush of the air stream into the nose proper. These prominences are formed by projections from the medial crura, the lateral crura, the caudal end of the upper lateral cartilages, the atrium (or cul-de-sac) between the upper lateral and lobular cartilages, and by a rim of soft tissue at the posterior end of the floor of the vestibule.

Resistance to the flow of air through the respiratory tract

is essential to good breathing; approximately 46 per cent of this resistance is encountered in the nose, of which the vestibule is a part. If there is too much space in the nose, or if there is so little space that breathing through the mouth becomes necessary, the effect of resistance is lost. All of you can remember some child with complete obstruction due to excessive adenoid tissue, in whom great improvement occurred as early as two weeks following adenoidectomy. Nasal obstruction can parallel such a case.

The functions of the septum are both anatomic and physiologic. The nasal septum creates two noses; this allows one side to rest (at least partially, according to Heetderks[10]) while the other takes over, as during sleep. The hard portion provides resistance to the air stream; it makes the dorsum and helps support the upper lateral cartilages and nasal bones. It is also the medial terminus for the rhythmic excursion of the turbinates, which assists in the control of velocity of air currents. The septum is part of the "upper lateral valve," separates the medial crura, and supports the respiratory epithelium; hence, there is a direct relation of the septum to all parts of the nose.

Injury to the external nose causing movement of the pyramid away from the midline is always associated with a septal injury, such as a dislocation at the caudal end or along the floor, or fractures varying from a single crack to multiple overlapping fragments. Also, injury or disease that causes movement, especially of the cartilaginous septum, in any direction, will produce some corresponding change externally. An example is "saddling," which may be due to a septal operation, injury, hematoma or abscess. Another example is the twisted nose due to the septum's being pushed over to one side.

Injuries can produce acceleration or deceleration of growth. It is not unusual to see an individual in whom examination of the lobule will enable you to determine the approximate date of injury. Fig. 1 shows a girl, age 16 years, who injured her nose before she was 10 years of age. Following the injury the lobule practically stopped growing; however, the region

above continued to grow, or even may have grown at an accelerated rate. It is also possible to have an accelerated septal growth pushing forward to such an extent as to cause a disturbance in the valve action, or a downward growth can produce what is called a hanging columella.

Fig. 1. Hump associated with an underdeveloped lobule due to injury.

A good classification of septal functional alterations is the one proposed by Cottle. He divided them into deviation, obstruction, impaction and tension. *Deviation* of the septum does not necessarily require surgical correction; however, if there is enough deviation in areas that cause trouble, operation is indicated. *Obstruction* implies intermittent blockage of the passage of air through one or both sides of the nasal cavity due to a pathologic septum. Treatment is frequently surgical correction. *Impaction* means that the septum is

pushed all the way over to the lateral wall, and movement of air in that area is completely blocked at all times. The condition is always treated surgically. *Tension* is due to an abnormal length or height of the septum, or many other factors which put stress and strain on the mucous membrane. This can be relieved only by reconstructing the septum to fit the other parts of the nose.

Reconstructive septal operations permit correction of all or any portions of the hard and membranous septum with avoidance of surgical trauma to uninvolved structures. It should be performed at the same time that neighboring structures which are associated with the deformed septum in disturbing nasal function are being corrected. The aim of this type of surgical treatment is to produce a good hard septum, a good membranous septum and a good semimembranous septum (columella). This will help preserve septal functions, prevent and correct saddling, prevent and correct retraction of a columella, and correct tensions due to conditions already described; moreover, the valve of the nose can be retained and may be "adjusted."

In all operations of this type we must keep foremost in our minds the relation of the upper lateral cartilage to the septum, for it is through the correct relationship of these structures that normal nasal respiration is maintained. There have been patients with short upper lateral cartilages in whom the valve of the nose has actually been "cut off" in the process of shortening the nose.

In addition to restoration of normal anatomic relationships, there are several other requisites of a successful septal operation. First, vertical incisions in the mucosa should be avoided because scarring in the nasal mucous membrane interferes with normal function; moreover, care should be exercised not to injure the perichondrium and periosteum; adequate exposure of all parts of the septum should be feasible and, finally, the incision should be easily approximated with sutures so as to permit healing by primary intention, and the relation of the septum to adjacent structures should be established. Cot-

tle's technique of opening the septum at its caudal end not only meets all these requirements but it is exceptionally safe when the combined septal operation and rhinoplasty are indicated (see Fig. 2); moreover, the Cottle incision permits repair of saddling of the cartilaginous dorsum, correction of a retracted columella, and performance of submucous resection of the upper lateral cartilage.

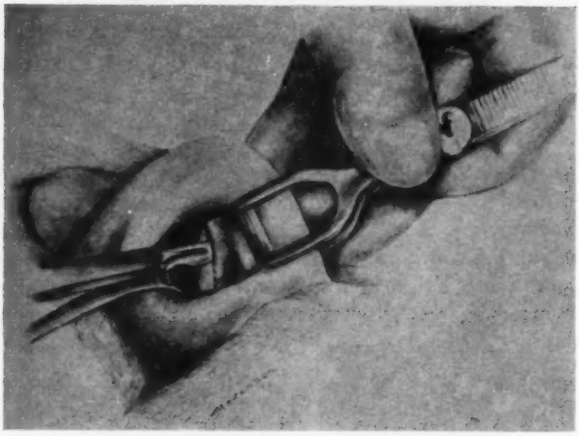

Fig. 2. Incision just above caudal end of septum. This is a hemitransfixion. (Picture by permission of Dr. M. H. Cottle.)

It is good practice to inspect the inside of an acutely injured septum when necessary and to manipulate the displaced parts under direct vision. Certainly, it is much less traumatizing than grasping the whole mucosa-covered mass with a forceps and blindly trying to reposition it.

Just as an acutely injured septum can be reconstructed, it is my contention that the same principles should be used in reducing or reconstructing an "old" septal injury. In such a septum naturally there would be no separate pieces of cartilage or bone; however, it is possible, after opening the septum, to make fractures or remove parts at the most advantageous places and thereby correct an abnormality with the least possible amount of trauma.

It is unsound merely to correct a septal injury when other abnormalities which affect normal functioning of the nose are present. Proper functioning may be affected by a number of conditions other than those already mentioned. For example, the long nose and drooping tip with nostrils facing on the lip cause the air currents to hit the top of the vestibule so that they cannot travel in their normal curve. A large hump can alter the eddying of the air. Dislocation of the caudal end of the septum and a depressed tip are frequently encountered, and septal surgery alone will not correct a flattened tip. Abnormalities of the alae, nostrils and columella disturb vestibular function, and their correction is the responsibility of the rhinologist. Proper direction of air currents, the intensity of flow, and the amount of air which may flow through the nose in a given length of time, provide different considerations to guide in the selection of patients for nasal surgical procedures and for deciding whether the patient needs an operation on the septum, lobule, external nasal pyramid, or any combination thereof.

In the patients upon whom I have operated, both for correction of a septal deformity and for the deformities of its associated structures, not only was improvement in physical well-being noted but in some a remarkable change also occurred in their mental outlook. Several illustrative cases will be cited.

REPORT OF CASES.

Case 1: Mrs. B. sustained a severe injury of the nose, causing an external deformity and a fractured and dislocated septum; the caudal end of the septum was in the right vestibule. Treatment consisted of opening the septum and reducing the septal fractures under direct vision. The pyramid was uncovered, the impacted left nasal bone was mobilized, and realignment of the bony vault was accomplished; the lobule was also operated upon. This is not a routine procedure, but a few months previous to this accident, this patient had consulted me in regard to nasal surgery. Probably the only difference in procedure was that at this time the osteotomy had already been done by the accident. Incidentally, with some shortening of the nose the apparent hump disappeared. A good functioning result was obtained.

Case 2: M. S., a German refugee, aged 26 years, had received nasal injuries while in many concentration camps. He had an impacted septum, a twisted nose and an overhanging tip. Reconstruction of the nasal septum and operation on the pyramid and lobule resulted in improvement in both nasal respiration and appearance.

Fig. 3. Case 1. Acutely injured external pyramid and septum.

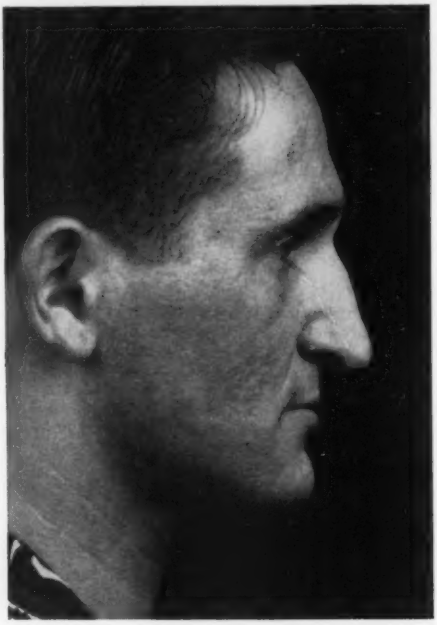

Fig. 4. Case 2. Old injuries including an impacted septum.

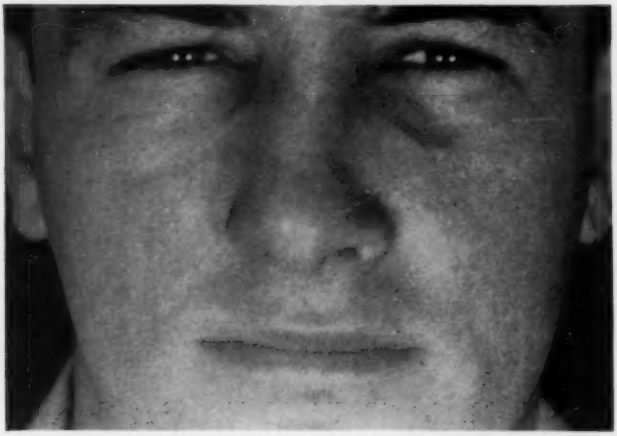

Fig. 5. Case 3. Acute injury causing a lateral displacement of the external nose and septum.

Fig. 6. Case 4. Obstructing septum bilaterally with tension forward.

Case 3: D. S., a professional football player, injured his nose while playing football, and it was "set" by the club physician. He was seen by me four days later. Not infrequently it is impossible to keep the external pyramid in proper alignment because the position of the septum has not been corrected; however, following opening of the septum and reduction of its fractures, the external nose was easily placed in the midline, and it has remained in that position.

Case 4: T. P., a professional baseball player, had an external deformity of the nose, an obstructing septum bilaterally and tension forward. Treatment consisted of removal of about two-thirds of the width of the septum by slicing sagitally, reducing tension of the septum by hump removal and lobule surgery. This resulted in practically normal respiration and considerable psychologic improvement.

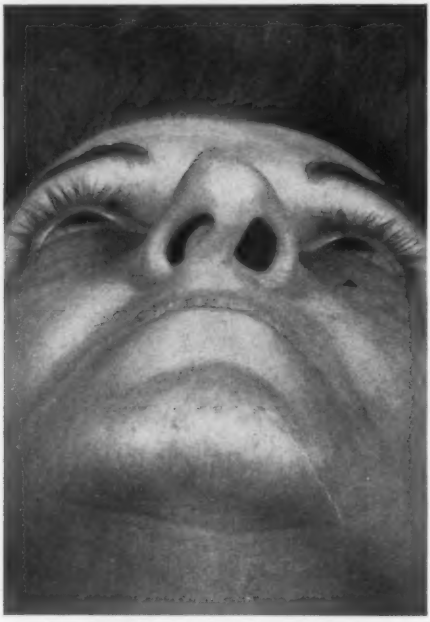

Fig. 7. Case 5. Hump, lateral deformity and obstructing septum caudally.

Case 5: Miss S. had a hump, lateral deformity of the nose and an obstructing septum caudally. At operation the septum was reconstructed, the hump was removed, the nasal pyramid was put in the midline, and the lobule was corrected. This resulted in improvement in breathing as well as appearance. This case emphasizes the futility of septal surgery without surgical treatment of other deformities of the nose.

CONCLUSIONS.

Septal reconstruction is a safe and physiologic procedure. It can be performed with or without manipulation of the rest of the nose. Correction of anatomic variations and restoration of the normal physiology should be our objective.

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TRACHEAL OBSTRUCTION FROM BENIGN LYMPHOID HYPERPLASIA IN THE CRICOIDAL AND TRACHEAL MUCOSA.

REPORT OF A CASE.*

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A two-and-one-half-year-old female child was referred to us for consultation regarding possible laryngeal pathology on Feb. 25, 1952.

The chief complaints were those of gasping for breath during exercise, a change in the voice and noisy breathing while sleeping. The family history was not pertinent. Past medical history revealed that the birth had been normal and that no respiratory difficulty had been noted. The mother stated that the baby had developed noisy breathing during the second week of life and that this gradually became worse. When the baby was three months old the parents became quite concerned and a pediatrician was consulted.

The general examination was reported to have been normal at this and subsequent times, with the exception of minor findings during acute upper respiratory infections. Chest X-rays in July, 1949, were said to have revealed enlargement of the thymus gland and three X-ray treatments were given at that time. A repeat X-ray in January, 1950, had shown the thymus gland to be of normal size. No significant improvement in symptoms was noted following the X-ray therapy. The baby was then given ACTH and later antihistamines without benefit.

In September, 1951, the parents first realized that the child gasped for air when she was running. As the child grew and

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became more active, her symptoms became more noticeable. In November, 1951, a squeaky and shrill tone was noted in the cry. Early in December, 1951, hypertrophic adenoid tissue was reported and three X-ray treatments were administered to the nasopharynx. The symptoms continued unabated and sleep became fitful.

At the time of our initial examination in February, 1952, the child was found to be well developed and well nourished, albeit somewhat apprehensive. The voice had a "tinny" sound

Fig. 1. Shows a marked narrowing of the trachea as it joins with the larynx.

which was accentuated while crying. Examination of the ears, nose, throat and nasopharynx revealed these structures to be normal. Definite respiratory distress was noted when the child was allowed to run around the office. Indirect laryngoscopic examination was not tolerated. A scout film of the upper airway revealed marked narrowing of the upper trachea as it merged with the larynx. The lumen of the trachea at this point was reduced to one-third of normal size by a mass arising from the posterior wall of the trachea.

The child was hospitalized and direct laryngoscopy was performed under a light general anesthesia. The larynx was found to be normal. A rounded, smooth, nonulcerated mass of firm consistency and with a broad base was seen in the posterior wall at the cricoid level. A 3.5 mm. bronchoscope could not pass the obstruction. The upper esophagus was examined and found to be normal. It was felt that biopsy specimens should not be taken until after a tracheotomy had been done, and the child was returned to her room.

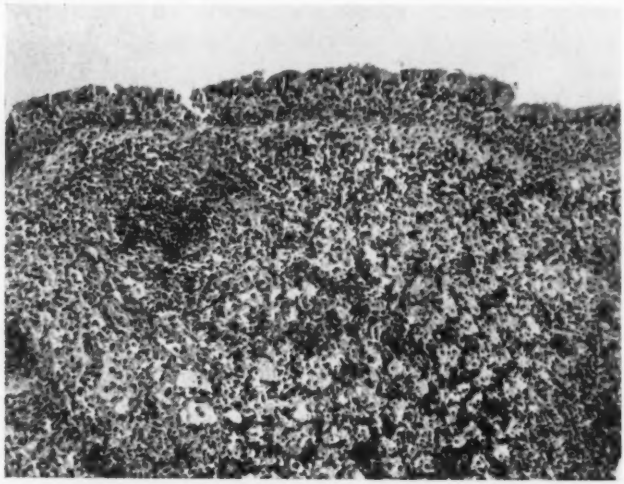

Fig. 2. The section is from the margin of the lesion. Low power. It shows a typical respiratory mucosa. There is moderate focal lymphocytic infiltration in the upper left corner, and a minimal diffuse infiltration elsewhere.

After consultation with a thoracic surgeon (Dr. Byron Evans) it was decided that a tracheotomy tube should be inserted as low in the trachea as possible, so that if it were necessary to resect the tumor, the trachea could be more easily mobilized.

The tracheotomy was performed on Feb. 29, 1952. The tube was inserted at the level of the seventh and eighth tracheal cartilages. A 5 mm. bronchoscope was then introduced and specimens of tissue were removed for microscopic exami-

nation. The child was returned to the ward in good condition and so remained until late that evening when her respirations became rapid and labored. A bilateral pneumothorax had developed, which necessitated thoracotomies and the insertion of a Foley catheter into each pleural space. The dyspnea promptly disappeared. The pneumothoracies cleared rapidly, and the Foley catheters were removed after three days.

Fig. 3. Section of mucosa and the mass. It shows respiratory mucosa of normal thickness, free from squamous metaplasia. Beneath the mucosa there is a dense infiltration of lymphocytes. Many swollen reticuloendothelial cells showing phagocytosis of cellular debris are present.

The pathology report by Dr. Lewis Palmer was as follows: "Sections through the small bits of tissue showed large numbers of closely grouped lymphocytes and a moderate number of reticuloendothelial cells in a fine reticulum stroma. Many of the macrophages contained abundant pale cytoplasm. In a few cells small numbers of pale pink-staining particles are found. One of the larger pieces of tissue is covered by respiratory mucosa of normal thickness. There is no evidence of malignancy."

The possibility of a nonspecific deposit of lymphoid tissue

was considered in the differential diagnosis, but it was thought that the biopsies might have missed a more deeply seated tumor, so specimens were removed on two other occasions. Following the last procedure, a 5 mm. bronchoscope could easily pass the biopsy-riddled mass. In reviewing all of the pathologic material and the clinical history, it was felt that the patient had a benign deposit of lymphoid tissue beneath the tracheal mucosa. The absence of ulceration of the mucosa and other microscopic features ruled out the possibility of a granuloma. The absence of squamous metaplasia of the

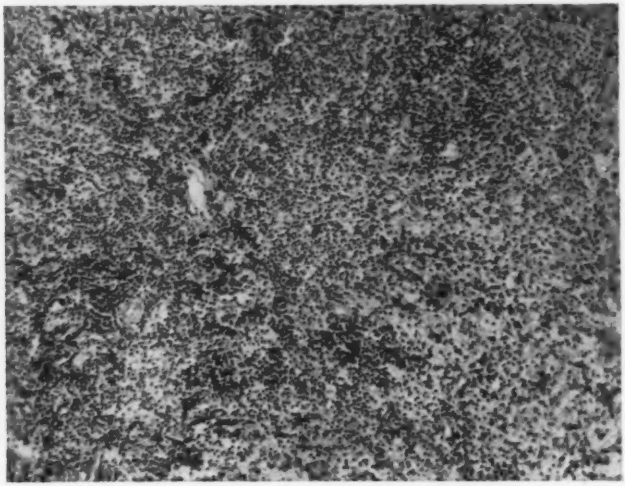

Fig. 4. The section is from a deeper level of the mass. It shows the dense grouping of lymphocytes, together with many reticuloendothelial cells.

mucosa in the first biopsy was considered evidence against simple chronic inflammation. The lesion appears to have been composed of densely grouped lymphocytes and many widely scattered macrophages showing active phagocytosis. The phagocytized material was cellular debris and neither histoplasma capsulotum nor fat.

The course from this time on was uneventful. Roentgenograms on March 15 showed that the mass had nearly disap-

peared. Tracheoscopy was done on March 18, and it was found that the tracheal lumen was normal in size, several low ridges remaining at the site of the lymphoid deposit. The tracheotomy tube was then removed. The child was discharged from the hospital 10 days later. She subsequently received a course of X-ray therapy of three treatments in 12 days, each of 125 r. of 200 K.V. filtered through 0.5 mm. of copper plus 0.5 mm. of aluminum over an area of 4 x 5 cm. The child has been seen periodically and seems to be normal in all respects.

Fig. 5. The mass noted formerly in the larynx has now nearly disappeared.

SUMMARY.

The subject of lymphoid hyperplasia is one of great complexity. The etiology ranges from acute and chronic infection through benign and malignant tumors to endocrine disturbances. This case is reported because of the unusual location of the lymphoid hyperplasia occurring in the absence of regional lymphadenopathy. The child had had respiratory symptoms since shortly after birth, which progressed insidiously. She had been under the care of a competent pediatri-

cian who had examined her frequently, and who at no time had found evidence of an enlarged spleen or liver, generalized lymphadenopathy or blood abnormalities. The serologic tests for syphilis and skin tests for tuberculosis and coccidioidomycoses were negative. The proponents of the staticolymphatic constitution concept might find some features to support such a diagnosis in this case; however, this patient has never had generalized lymphadenopathy or signs of hypoadrenal function. In addition, she has manifested a superior constitution by being able to survive respiratory infections, despite marked tracheal obstruction.

CONCLUSIONS.

The trachea normally contains a few small deposits of lymphoid tissue in the submucosa of its posterior wall. This case is an example of an unsually large lymphoid deposit which had grown in response to minor infections in the upper respiratory tract. Endoscopic removal of the tumor appears to have produced a clinical cure, but only time will tell whether or not subsequent infections will bring on a recurrence of the mass.

I thank Dr. Carl V. Weller, of the Department of Pathology of the University of Michigan School of Medicine, for reviewing the biopsy material in this case.

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CLIMATOLOGIC FACTORS IN EAR, NOSE AND THROAT DISEASE.*

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The entire human organism is immersed in an ocean of air, affecting every physiologic function. The influence of weather and climate is perhaps most pronounced in the upper respiratory tract which communicates directly with the surrounding air. It may thus be of interest to review the rôle of climatological factors in causation and treatment of ear, nose and throat diseases.

Most of the bodily changes due to climatic factors are promptly compensated by normal physiologic processes. Climatic conditions, therefore, become a medical problem only when the demands exceed the ability for spontaneous adjustment. The individual always responds to weather and climate in a setting of physical and social circumstances, including occupational status, housing, diet, sleep, rest and recreational activities. His reactions are modified by personal makeup as well as past and present life experiences, and entirely heterogeneous causes can produce the same type of stimulation. Only on the basis of a detailed history and evaluation of all relevant data will it be possible to gain a diagnostic impression of the probable cause of repeated diseases of the upper respiratory tract. No patient should be labelled as meteorosensitive simply because his attacks coincide with abrupt changes of weather or the inclement season of the year.

The complexity of the biologic response is only one of the difficulties encountered in the evaluation of the influence of weather and climate on health and disease. Atmospheric con-

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ditions are a combination of various elements, such as temperature, precipitation, humidity and air movements. Modern meteorologists (weather men) take other more specialized factors into consideration and have at the same time introduced wider concepts, particularly those of air masses and air fronts, which are very useful for a better correlation of meteorologic cause and biologic response.

In the following review of the influence of weather and climate on ear, nose and throat conditions the emphasis rests on clinical experience. The climatologic component of the various upper respiratory diseases is examined; the main types of regional climates are described before evaluating the merits of climatotherapy; and finally the influence of air-conditioning is briefly surveyed.

INFLUENCE OF WEATHER AND CLIMATE ON VARIOUS EAR, NOSE AND THROAT CONDITIONS.

It is generally assumed that no other disease is more immediately related to weather than the common cold. To be sure, the term "common cold" does not refer to a strictly circumscribed pathologic entity but is used to cover all acute respiratory infections, be they due to invasion of viruses or bacteria, or occurring on an allergic basis.

It has not yet been possible to assess with certainty the degree to which the common cold is exclusively brought on by changes in weather and climate. Entirely different stimuli, as for instance meteorologic conditions, especially lowering of temperature and chilling drafts; psychogenic factors; reduced vital resistance, and various allergens, may have identical effects upon the upper respiratory system. Thus it is often almost impossible to determine to which cause the onset of disease should be attributed in a particular instance.

Among the factors which are apt to upset the equilibrium of the sympathetic nervous system, *sudden chilling* should be mentioned first. Subjects vary greatly in their response to chilling, some being affected by cold air but not by contact with cold water or ice, while in others the opposite is the case.

Again, among those reacting to cold air, sensitivity may depend upon the degree of air humidity, and some experience chilling from damp but not from dry cold air.

Chilling of body surfaces indicates a disturbance of the heat balance of the human organism, and occurs at least as frequently during the warm season as in winter. With higher degrees of outside temperature the body is covered with a film of sweat and the skin vessels are dilated, while the ability to fight off infections is greatly reduced.

Draft in itself is not the cause of chilling, and colds due to drafts are in reality the result of insufficient accommodation, or a sign of pre-existing disease. Strong winds, drenching rains, snowstorms or even subfreezing weather are less likely to bring on chilling than a slight air current coming in spurts, or sudden and repeated transitions from protective warmth indoors to lower outside temperatures, requiring continuous adjustment of the capillaries to environmental conditions.

The common cold can be regarded as a crisis in the adaptation to changing atmospheric conditions, the reaction depending upon the ability of the organism to offset the outside stimulus through an adequate and balanced response. It is of little importance whether the change is due to transition from one season to the next, or from one room to the next. Similarly, it is irrelevant whether the repetition is brought on by successive cold fronts or by moving from indoor to outdoor temperatures. In addition to such stimuli derived from weather and climate, however, the autonomic nervous system is also subject to a number of other influences which may reduce its ability for adequate adjustment.

Vasomotor rhinitis represents a massive response of the autonomic nervous system to environmental stimulation, and has been traced to sympathicotonia, *i.e.*, domination of body functions by the sympathetic nervous system. Various conditions may bring on complete blockage of the nasal passages, often combined with paroxysmal attacks of sneezing, or secretion of thin mucoid discharge. Patients afflicted with vasomotor rhinitis may experience attacks when the weather turns

cold, the air humidity increases, when it becomes sweltering and oppressive, or again with low air humidity, or wind. In subtropical climates episodes of vasomotor rhinitis are observed when sudden cold winds strike while the sun is shining brightly and the body is still adjusted to the comfortable warmth.

Atrophic rhinitis is closely linked to excessive heat and the degree of air humidity. The incidence is especially high in the hot dry air of artificially heated buildings, which tends to reduce the water content of the respiratory tract.

The influence of weather and climate on the earlier stages of *sinusitis* is well established. Infection of the accessory sinuses is prevalent in the northern temperate zone during winter, but develops also in the hot, dry and often dust-laden air of arid and semiarid regions. While chronic sinusitis is not directly related to atmospheric conditions, the incidence of exacerbations is much higher in an unfavorable environment.

Epistaxis may assume almost epidemic proportions at times of abrupt weather changes. The atmospheric factor is clearly evident in epistaxis due to "thinning of air," as, for instance, in mountain sickness.

Among diseases of the mouth, *Vincent's angina* is decidedly subject to seasonal influences. The tonsils, often a seat of latent infection, are affected by abrupt weather changes, and the result may be *acute tonsillitis*, peritonsillar abscess or "sore throat." Croup and laryngitic involvement of the throat are more common in late winter and early spring colds, which are usually repeat attacks of upper respiratory infections, while as a rule in the Fall only the nose is affected.

Eustachian catarrh is frequently a secondary manifestation of sinusitis, and is subject to the same causes of environmental stimulation. *Mastoiditis* often develops following a drop in air temperature, or when a second cold front strikes before the organism has had time to recover from the impact of an earlier meteorologic disturbance.

Physiological exhaustion is most pronounced just before the advent of Spring. Thus *septic sore throat, scarlet fever* and *epidemic meningitis* reach their highest incidence in March, while respiratory diseases, especially cold, influenza and pneumonia are most common during the month of January. Due to improvements in hygiene and medical care, and especially since the introduction of sulfa and antibiotic therapy, the contour of mortality curves may no longer be representative of the seasonal character of certain diseases. On the other hand, preseasonal immunization against catarrhal illness, while reducing the overall incidence of colds, does not affect the pattern of seasonal distribution of upper respiratory infections.

ALLERGY.

Abnormal sensitivity to weather conditions may assume the characteristics of an allergic reaction. But the particular allergy to cold and meteorologic disturbances often takes the form of rhinitis and other marginal affections of the autonomic nervous system. There seems to exist certain autonomically unstable or hypersensitive persons who react to normally innocuous stimulants, be they of a meteorologic or of a strictly allergic nature.

The question arises whether truly allergic subjects are affected by weather and climate to a higher degree than other persons. Even in patients with a history of seasonal allergy of several years' standing, this point can be decided only on the basis of a detailed study, always taking into consideration the possible influence of other causes. The drying effect of desert air is conducive to hay fever, while in any climate chilling may bring on recurrences of the disease.

AIR POLLUTION.

An atmospheric factor which during recent years has assumed increasingly greater importance is air pollution. With progressing industrialization the air is more extensively abused as a dumping ground for unwanted airborne materials. Ordinarily, the highest degree of contamination is

reached between sunrise and noon, and conditions are worse after the passage of a high-pressure area, when there is warm air aloft, with light winds and no precipitation.

The exact effect of smog on ear, nose and throat is still a controversial subject, but it has been definitely established that the incidence of common respiratory diseases is generally higher in congested work areas with multiple industrial operations. Morbidity due to the common cold, chronic bronchitis, hay fever, sore throat, acute sinusitis and cough is greatest in the age group of 55 years and over. This increased proneness of elderly people to upper respiratory diseases demonstrates that an atmospheric factor—in this case polluted air—may assume importance only after the organism as a whole has been weakened through natural processes.

MICROCLIMATE.

Weather and climate show sometimes a considerable measure of variability in circumscribed areas, and it is always advisable to study in detail the specific microclimatic conditions under which the patient lives and works.

Metropolitan areas may extend through several climatic regions, and fluctuations of the different meteorologic factors often show extreme values. High buildings interfere with proper air circulation, and the pall of smoke and dirt prevents cooling by irradiation. Cloudiness and precipitation are increased, but the relative humidity is lowered as the water is drained off as rapidly as possible, and only a small part is available for moistening the air by evaporation.

Not only the location of the home but also conditions within the house itself are of climatic importance. The cellar is humid, with a relatively constant temperature all year round. Variability increases from floor to floor, and often reaches a maximum under the roof. Even within one single room the differences may be considerable. Thus the feet of a person are often exposed to a different climate than the head. By the same token, small children live under other climatic conditions than adults.

CLIMATOTHERAPY.

Changes in the microclimatic environment can well be regarded as a part of climatotherapy. Up to the present time, however, this term was mainly reserved for the removal of a patient to a different climatic region. A more suitable atmospheric environment may contribute to the control of chronic diseases of the upper respiratory tract and prevent new attacks which could complicate systemic illness.

In the temperate zone the moist *ocean air* has a beneficial effect on atrophic rhinitis. Upper respiratory infections become less frequent, are milder and of shorter duration.

Mountain climate offers lower humidity and lower pressure, combined with increased ultraviolet radiation. Higher altitudes are healthful only for indiduals with a more vigorous constitution, while others will do better in the milder and more protected valleys or foothills.

Patients subject to recurrent upper respiratory diseases experience the greatest relief in *southerly latitudes*. Even a temporary sojourn affords respite from the constant fluctuations of passing air fronts.

The climate of the entire Southwest is characterized by almost perpetual sunshine, pure air, low humidity, and something approaching complete absence of storminess. Within this climatic region there occur considerable variations, mainly due to differences in altitude and vegetation.

Freedom from meteorological and climatic crises is of special benefit to aged persons and thoroughly exhausted children, but physiologic adjustment to a constantly warm environment is not complete for many months. Consequently a trial period of at least 12 months is advised before deciding upon a permanent domicile.

Desert climate is not suited for every patient. The wind often carries considerable dust and sand, which may be as irritating to the upper respiratory system as fog along the coast. The typical complaint of "dry nose," however, is effec-

tively treated by oil nose drops or sprays. Quite generally, irritation of the nasal mucosa must be avoided in the dry climate of the Southwest, and if aqueous solutions cannot be dispensed with they are followed up by an oil spray or oil drops.

In the Southwest with its low ragweed pollen content, the allergic subject is relieved of the attacks he used to suffer at the place of his former residence. The region, however, is by no means entirely without its own allergens, and these less desirable aspects of the Southwest are mentioned in order to obviate the impression that it is the Garden of Eden, pure and simple, without any of the drawbacks of life terrestrial.

ADVISABILITY OF CLIMATOTHERAPY.

Climatotherapy is not always indicated, and the decision depends in each instance upon the clinical judgment of the physician. Often gradual adaptation to an unfavorable environment can be achieved. On the other hand, even a change to a more desirable climate may bring on infection, because the patient is subjected to new conditions, new air currents, new bacteria against which he has not yet developed any defense.

Many a person has found it more difficult than anticipated to become accustomed to a strange environment, particularly at a time when he was physically least equipped to do so. Faced with the problem of finding a new means of livelihood and making new friends, he may experience recurrences or exacerbations even under more desirable climatic conditions. When the threat to a person's security becomes oppressive, even an ordinarily healthy subject may develop any number of upper respiratory disturbances.

All these observations, however, should not detract from the medical value of climatotherapy. Though climatic conditions represent but one segment of the patient's total environment, it may under certain circumstances be the only one amenable to change. A more equitable climate affords biological rest, which is important for elderly people and patients

of *any* age suffering from chronic conditions, which might be complicated by recurrences and intercurrent infections. On the other hand, little improvement can be expected when upper respiratory diseases have advanced to the stage of irreversible anatomic changes.

The benefit of climatotherapy depends largely upon correct timing. While it is preferable to start the trip at the beginning of the inclement season, any shortening of exposure to cold and meteorologic disturbances is of advantage. More lasting results are usually obtained if the patient delays his return home until atmospheric conditions there have become at least moderately settled.

AIR-CONDITIONING.

Modern man has succeeded to a certain degree in changing climate itself. Particularly, air-conditioning has not only made hitherto uninhabitable regions accessible, but has also greatly contributed to health and comfort in the moderate latitudes. Yet, the man-made climate of air-conditioned buildings has also created new problems.

Cooling for comfort in warm weather is a far more intricate problem than heating in winter. Individual response to temperature varies to such an extent that it is almost impossible to balance divergent requirements and to establish conditions which are generally satisfactory. With extreme summer temperatures best results may be expected from lowering of air humidity to 40 or 50 percent, combined with cooling to a level about 10° F. below outside readings. Greater differences in the temperature level may cause sudden chilling, colds and other respiratory infections, especially in movie theaters, office buildings and air-conditioned airplanes and trains.

An important aspect of air-conditioning is purification of the circulating air. The incidence of airborne infections is reduced by suppressing dust and decreasing the bacterial content as well as the rate of allergens. Thus efficient air-conditioning has a protective influence on persons living or working in a controlled atmosphere.

CONCLUSIONS.

At the present state of medical knowledge the physiology of climatic response does not lend itself to comprehensive generalizations. Additional observations and continued research may be expected to throw new light on established facts, and promote a better understanding of the effect of weather and climate on the upper respiratory tract.

These influences, to be sure, constitute merely a borderline problem of otolaryngology; but the physician is almost daily confronted with evidence of the impact of weather and climate on ear, nose and throat diseases. Atmospheric conditions should always be taken into consideration in compiling the patient's history, as such information may prove of aid in arriving at a diagnosis and in adapting the standard procedures to the requirements of a particular case.

Modern climatotherapy is a highly flexible adjunct of otolaryngology. When selected in accordance with the particular needs of the patient it will not only alleviate suffering but also aid in the recovery of lasting health and fitness.

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CHANGING CONCEPTS REGARDING THERAPEUTIC MANAGEMENT OF CERTAIN ENDOSCOPIC PROBLEMS.*†

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Endoscopic practice continually presents many interesting and at times disheartening problems. Most of these can be satisfactorily handled and good results secured by endoscopic techniques. In fact, many of these can be handled *only* by the endoscopist; however, others at times, by the very nature of the pathology involved, present no lasting relief or cure of the condition by such means, and eventually other methods outside of endoscopic practice must be called upon. Until comparatively recent years due to the limitation of surgical methods available, no surgery could be attempted in these cases, and endoscopists would be called upon to maintain the long-time treatment, which, although not at all ideal, was the best available. This long-continued treatment, which in some cases necessitated many operative measures, was at best very trying to both the patient and the endoscopist. With the marked improvement in the techniques of the thoracic surgeons, consisting not only in those of the operating surgeon but also in the realm of anesthesia and in the method of combating infection, we endoscopists have been relieved of some of the burden and the patient restored to a more normal method of living.

It is with this thought that we who practice endoscopy should be alive to the possibilities now offered by thoracic surgery, that I venture to call attention to the three types of cases in which at times we and the patients have been sorely

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distressed by chronic or recurring conditions for which we have no cure, but only palliation obtained by a long-continued treatment or multiple operations. These three types of cases may now be considered to be suitable candidates for remedial surgery, with a fair hope of success and without too much danger to life.

We shall, therefore, consider:

1. Surgical remedy for persistent achalasia of the cardia which has not responded to a reasonable degree of dilatation by endoscopic means.

2. Cases of severe strictures of the esophagus which do not improve sufficiently by the usual methods of dilatation, therefore necessitating long-contiued treatments.

3. Benign lesions of the trachea and the upper portion of the main bronchi, such as adenoma, which are either too far advanced or too deeply penetrating in the wall of the organ to prevent complete removal by endoscopy and which have in the past been considered beyond surgical attack.

Cardiospasm probably accounts for about 20 per cent of all cases of dysphagia. It occurs about twice as often in females as in males and may cause symptoms from a very early to an advanced age. As a rule, the symptoms are most frequently met in the third or fourth decade, but many of the patients who present themselves have had intermittent dysphagia for several years before consulting a physician.

Thomas Willis,[1] in a book published in 1679, gives this eloquent description of what must have been a case of cardiospasm:

"No less well, a very rare case of a certain man of Oxford shew an almost perpetual vomiting to be stirred up by the shutting up of the left orifice. A strong man and otherwise healthful enough, laboring for a long time with often vomiting, he was wont very often, though not always, presently to

cast up whatsoever he had eaten. At length the disease having overcome all remedies, he was brought into that condition, that growing hungry he would eat until the oesophagus was filled up to the throat, in the meantime nothing sliding down into the ventricle, he cast up raw (or crude) whatsoever he had taken in: when that no medicine could help and he languished away for hunger and every day was in danger of death, I prepared an instrument for him like a rod of whale bone with a little round button of sponge fixed to the top of it; the sick man having taken down meat and drink into his throat, presently putting this down in the oesophagus he did thrust down into the ventricle, its orifice being open, the food which otherwise would have come back again; and by this means he hath daily taken his sustenance for 15 years and doth yet use the same machine and is yet alive and well, who would otherwise perish for want of food. Without doubt in this case the mouth of the stomach always closed, either by a tumor or Palsie, nothing could be admitted into the ventricle unless it were violently opened."

Mikulicz,[2] about 70 years ago, used the term "cardiospasm" in designating a group of cases presenting dysphagia and dilatation of the esophagus. He assumed the condition to be due to true spasm of the lowest segment of the esophagus. Meltzer, and later Hertz,[3] expressed the belief that the condition was brought about by the failure of relaxation of the lower end of the esophagus during the act of swallowing. These two points of view have been debated ever since and to my knowledge no definite proof has been shown that either is the sole cause in all cases.

It has been commonly observed that many patients have symptoms for years without a great dilatation of the upper esophagus, while other cases present megaloesophagus of a marked degree. It has also been noted that in cases of organic obstruction, such as those due to stricture, only a slight degree of dilatation occurs and that, immediately above the obstruction. For this reason many observers believe that both spasm of the lower esophagus and atony of the upper esophagus may each have a part in the causation of this condition.

Rake[4] first observed the decrease or absence of Auerbach's plexus of nerve cells in cases of megaloesophagus and these observations have also been made in cases of Hirschsprung's disease of the colon. Puppel[5] devised an ingenious method of measuring the motility and peristaltic action in cases of megaloesophagus by means of an inflated balloon placed in that organ and registering the esophageal efforts at expulsion by air pressure registered on graphs. He concluded that, "A disturbed peristalsis or even total motor paralysis of the whole esophagus may be present in this disease."

In the medical and surgical therapy of these cases we must, therefore, take both of the above proven factors into consideration. When we who practice endoscopy see these patients for the first time, they have run the gamut of the usual medical therapy, generally consisting of dietary measures plus the use of antispasmodics. These patients as a rule, for a while, respond favorably to the medical regime, but recurring episodes of dysphagia have in time responded less well until the patient is very distressed both physically and mentally. They are then treated by various methods of dilatations, and as a rule for a short period may be completely relieved by the ordinary stretching of olive bougies over the guiding string. Even the passage of a mercury-filled Hurst dilator occasionally suffices for a time. Eventually, and generally quite soon, a more efficient method is demanded. This consists of a very forceful dilatation by means of a distensible bag placed in the cardia and expanded by air or water. We have all used these procedures with undoubtedly a great deal of satisfaction in most cases, although I, personally, upon myself, would prefer surgery to forceful dilatation.

Olsen *et al.*,[6] at the Mayo Clinic, reported in 1951 that about 80 per cent of the patients were relieved by forceful dilatation. Maingot[7] estimates that the proper use of the above method will cure about 70 per cent of all cases and give improvement to about 20 per cent. There remains about 10 per cent of all those treated in whom such methods fail. I would suggest that among the 20 per cent of the cases improved

there are probably many who would prefer "getting the job over," if a good prognosis with reasonable safety could be assured by the thoracic surgeon.

The advances made along these lines in the past few years have now not only justified our recommending surgery to those whom we have failed to relieve, but also to present it for consideration to many of those who have had only moderate or temporary relief from our efforts.

Surgical efforts along these lines have followed various hypotheses of the etiology of the disease. The method of manual dilatation through a gastrostomy as advocated by Mikulicz[2] has long ago been given up. Likewise, the operation to remove the sympathetic nerve supply to the cardia has been rejected. Surgical intervention has now been restricted to one of the following methods:

1. Cardioplasty: Advocated by Wendel, in which a longitudinal incision through all layers of the lower esophagus is made and closed in a transverse manner.

2. Esophagogastrostomy: Advocated by Heyrovsky and later modified by Gruendahl.

3. Extramucosal Myotomy: First developed by Heller,[8] consisting of a longitudinal incision through all muscle layers to the mucosa along the anterior wall of the esophagus and extending down into the stomach.

4. Wangensteen Procedure: Consisting of the lower portion of the dilated esophagus and the acid producing portions of the stomach followed by anastamosis of the remainder of the stomach and esophagus. At the same time a Ramstedt type of pylorotomy is performed. The excision of the fundus of the stomach prevents future acid regurgitation into the esophagus and the Ramstedt compensates for the vagotomy which necessarily occurs during the main operation.

It is not the purpose of this paper to discuss the operations from a technical standpoint. It would seem, however, that time may prove that the Heller type of operation may be the

most desirable as a general rule, although the Wangensteen may have a place especially in cases of severe megaloesophagus. Barrett and Franklin,[9] reporting a series of late results in cases operated by esophagogastrostomy and cardioplasty, have pointed out that these cases often develop esophagitis due to the removal of the mechanism at the cardia which normally should prevent regurgitation of acids into the esophagus. Maingot[7] reports a series of 39 cases of Heller operation with excellent results in 38. The Heller operation is a much more simple procedure than any of the others, and the mortality much less; what is probably most important, it does not destroy the natural barrier of gastric juices to the esophagus.

We have collected the figures on cases operated upon in Buffalo by five different surgeons in four hospitals. Twenty-four cases of cardiospasm resistant to dilatations have come to surgery. Of these, 12 have had a modified Heller operation. Of these, two have been lost sight of, and 10 have been followed. Of these 10, nine had excellent results over a one- to three-year period since their operations. One has had an excellent result so far, having been operated upon three months ago.

In 12 cases of esophagogastrostomy, again, two cases have disappeared from view and 10 have been followed. Of these 10, four have had excellent results and one only a fair result. Five have been failures, as manifested by dysphagia and severe esophagitis with bleeding, which was undoubtedly due to acid regurgitation.

This is not an extensive list, but would seem to bear out the thought of many surgeons today, namely, that the modified type of Heller operation is the preferable one in uncomplicated cases of persistent cardiospasm.

The following is a typical case history:

Mrs. V. G., age 30, was first seen on Nov. 10, 1947, with the complaint of complete dysphagia. She was a rather nervous, active, somewhat emotional woman, who first presented herself at my office with the complaint of being unable to eat or drink for the past two days following the ingestion of steak which seemed to stick in the region of the lower esophagus. She stated that she saw her family doctor, who gave her belladonna and later attempted to pass a stomach tube without success. Her

past history was practically negative except for that of slowly progressive dysphagia for the past three years. She stated that about three years ago she began to experience difficulty in swallowing food only occasionally, but that this difficulty had become more frequent and within the last few weeks at every supper and nearly every lunch, food, especially the coarse types, such as meat, dry biscuits, etc., seemed to stick in the lower esophagus, and she had to wash them through with water. No food stuck for any great length of time until two days ago when she was eating steak, and she felt a piece stick in the esophagus, which she was unable to dislodge with water and which was not affected by the belladonna or the stomach tube which were used by her family doctor.

The patient was admitted to the Buffalo General Hospital on Nov. 10, 1947. Physical examination and laboratory work produced no significant findings. Under sodium pentothal a 9 mm. esophagoscope was passed and it was noted that in the lower esophagus there was a large, firmly impacted bolus of meat in the region of the cardia. This was removed by forceps, and it was further noted that there was a considerable degree of esophagitis, superficial ulceration and free bleeding. Four days later this patient was again examined by esophagoscopy and at this time there was no pathology in the esophagus or the cardia. X-rays taken on Nov. 14, 1947, showed no pathology whatsoever in the esophagus. The patient was discharged from the hospital and came to my office for dilatations of the esophagus from time to time, which gave her considerable relief.

On July 13, 1948, she again reported with the complaint of complete dysphagia for two days. She stated that again her trouble was caused by a piece of steak which was stuck in the lower esophagus. She was again esophagoscoped under sodium pentothal anesthetic, and at this examination it was noted that there was considerable esophagitis and spasm in the area of the cardia. The previously ingested steak had evidently passed under the effects of the anesthetic. The patient was again discharged and told to report from time to time for dilatations. These dilatations, however, were not carried out satisfactorily due to the patient's dislike for them and her nonappearance as directed.

On June 7, 1949, she was again admitted to the hospital with dysphagia of two days' duration. The patient stated that for the past year she had had considerable trouble with food sticking at the cardia, and that she had had a great deal of pain and heartburn, which did not respond to the ministrations of her family doctor. Fluoroscopic and radiographic studies of the gastrointestinal system taken on June 8, 1949, showed no pathology whatsoever in the esophagus, stomach or duodenum. Esophageal activity appeared normal, and the patient was discharged back to the care of her family doctor.

On May 31, 1950, this patient was again admitted to the hospital with the complaint of meat sticking in her lower esophagus, causing complete dysphagia of two days' duration. Esophagoscopy at this time showed a large bolus of meat firmly impacted at the cardia. This was removed, following which a ballooning gastroscope was passed into the stomach. There were no strictures, ulcerations or appreciable spasm. The patient was again referred back to her family doctor.

In September, 1950, this patient presented herself again and stated that for the past year she had had more difficulty in swallowing, that food stuck at the cardia, and that she had had considerable pain. She stated that of late she was practically never free of these symptoms. She further stated that the complete dysphagia seemed to pass after several

hours of waiting, but that she suffered a great deal of distress at her meals and had to eat very slowly and regurgitate at frequent intervals. She had now become not only distressed but depressed and felt that she could not dine out with her friends because she never knew when she would have to leave the table to regurgitate. She had lost 15 pounds in weight, although she had always been a rather thin woman. She also stated that she worked and this condition bothered her there, because she was afraid that while working she would regurgitate. Inquiries into her home conditions showed no major cause for mental trauma. At this visit we explained to her the possibilities of surgical intervention for this condition and recommended that she enter the hospital for such an operation. She readily consented as her condition had slowly progressed over three and one-half years to the point where she felt that life was no longer worth living.

On Sept. 19, 1950, she underwent a Heller operation for correction of achalasia of the cardia. A modified Heller operation was done consisting of an incision throughout all the muscular coats of the esophagus beginning about three and one-half inches above the stomach and extending down the stomach about two and one-half inches. It was noted by the surgeon that both the longitudinal and circular muscular fibres of the esophagus were very thick but there was no fibrosis. He also noted that there was bulging of the esophageal and stomach mucosa on the edge of the incised muscles throughout the entire length of the incision, causing gaps in some places fully an inch in width.

This patient made an uneventful recovery, and she has been observed now for over two years and has been free from all dysphagia and pain except during the past month when she had a return of very mild symptoms. These were cleared by the passing of one large mercury bougie. The patient has gained weight, her general outlook on life has become entirely different, and all in all she has had excellent results from this operation.

It would seem to me that we endoscopists should probably adopt a more surgically minded viewpoint and consult with the thoracic surgeon much sooner about problem cases. Wangensteen[10] has made the query, "Why persist in attempting to effect myotomy by nonoperating means if a few dilatations do not cure?" Wangensteen states further in closing the discussion of his paper, "It needs to be emphasized that a rather large number of patients are being committed to semi-invalidism in the outpatient clinics by being treated interminably by the passing of bougies by the otolaryngologist. If one makes an attempt or two at effecting bloodless rupture of the muscle fibres at the distal end of the esophagus by use of a hydrostatic dilator and does not succeed, I would say a Heller operation of myotomy is in order. It must not be forgotten either, that a bloodless myotomy achieved by use of the hydro-

static dilator is not without risk." I can subscribe to the latter statement as I have had a fatality caused by a minimum of stretching.

For many years the standard treatment of strictures of the esophagus has been repeated dilatations. These generally have been accomplished either by direct vision from above through an esophagoscope or by passing a dilator over a guiding string. Recently Tucker[11] has devised an indwelling dilator which gives promise of hastening the treatment. When the above methods have failed, endoscopists have tried the more effective method of retrograde dilatations through a gastrostomy. These treatments by dilatation have as a rule proved quite effective; however, in certain seriously strictured cases they have either failed, or demanded so many treatments that the time required, the frequency of anesthetics, and the expense have been a very great burden, not only on the patients but on their families as well.

For several years, surgeons have experimented on methods that would be satisfactory and not require such long care. Operations involving the esophagus were considered extremely dangerous for the following reasons:

1. Lack of adequate blood supply.
2. Lack of a protecting serous coat and omentum to seal off the suture lines.
3. Inaccessibility of the mediastinum.
4. Lack of firm submucosa for the sutures to grasp.
5. Difficulty of avoiding tension on the lines of suture.

Research on dogs by Macmanus *et al.*,[12] by Scheck *et al.*,[13] and Kleinsasser *et al.*,[14] among others have shown that the vascular supply of the esophagus is not so poor as had been thought and that the whole organ may be freed from its blood supply for its entire length and still remain viable.

At first, new passages to replace the esophagus were made by skin tunnels connecting the upper esophagus and the

stomach. Later, loops of gut were used with some success. These methods now have become obsolete due to the success of the procedures developed by Sweet[15] and others. These consist of subtotal resection of the esophagus and anastomosis of the free upper end of the esophagus with the stomach, which has been mobilized and brought up into the thorax.

The remainder of the problems were merely those of technique and these have been mastered, so that the operations involving the esophagus are now routine affars in the hands of the thoracic surgeons.

There yet remains one type of stricture to be considered. This is a very firm stricture, short in length, but, due to the depth of the original lesion and also late treatment, has marked scarring and contracture and has proven refractory to dilatation, unless these are repeated at short intervals and over a long period of time.

Swanson and Clatworthy,[16] Parker and Brockington[17] and others have shown the possibilities of end-to-end anastomosis of the esophagus in dogs. It has also been shown that in cases of congenital atresia of the esophagus that many times an end-to-end anastomosis of the esophagus may be made in these cases. Holt *et al.*,[18] at the Boston Children's Hospital, have reported 26 cases of survival following this method. Recently Gross[19] reported the successful end-to-end reconstruction of the thoracic esophagus of an 11-month infant by resection of a strictured segment, 3 cm. in length, and the end-to-end anastomosis of the esophagus. This patient made an uneventful recovery and had no further dysphagia.

In the past two years we have had two cases which we considered suitable for surgical intervention because of the great extent of the strictures and also the impossibility, at least in our minds, to give normal swallowing function. Both cases had a subtotal esophagectomy after the manner of Sweet and perfect results were secured.

N. H., age four years, was admitted to the hospital with the chief complaints of dysphagia of seven weeks, and a weight loss of 19 pounds in that time.

This child was well until seven weeks previously, at which time she drank lye settlings. Her grandmother gave her egg white and she drank water. She was taken to a hospital in her home town and X-rayed, then taken home again for a three-week period, without treatment. She was then taken to a hospital in another town for 12 days and remained there without dilatations until she was taken to the Buffalo Children's Hospital on Oct. 14, 1945.

For the first three weeks after the accident she was able to swallow approximately three glasses of milk or eggnog each day. Then she began having emesis after each swallowing. The character of the vomitus at this time was that of undigested food. It seemed as if it had not even reached the stomach. She began having nocturnal emesis of thick, mucoid, greenish or yellowish material. If the child was quiet and not nervous and nothing happened to excite her, she was able to swallow fairly well. The doctor had prescribed Syntropan, one-half hour before she ate. This medication seemed to help at first, but she soon became worse and could eat nothing.

X-rays taken at the first hospital showed a funnel esophagus.

Physical examination showed a well-developed, markedly dehydrated, four-year-old, white female child with evidence of recent weight loss and appearing chronically ill. The remainder of the physical examination revealed no significant findings. Laboratory findings were negative except for a considerable degree of secondary anemia. Fluoroscopic and radiographic examination of the esophagus with thin barium showed a slight dilatation of the upper esophagus which narrowed smoothly, becoming considerably restricted at the level of the fifth dorsal vertebra. The esophagus from this point to the cardia was distinctly narrowed with the mucosa slightly roughened. Some of the thin barium, however, did pass into the stomach.

On Oct. 15, 1945, the child was esophagoscoped. Esophagoscopy showed a stricture at the upper end of the esophagus. By passing a small bougie we definitely felt two more strictures: one, the tightest, being just above the cardia. All strictures were dilated to No. 17 French. Evidently the lower stricture had considerable granulation tissue, as free bleeding resulted. The final diagnosis was stricture of the esophagus.

After the child was dilated she was put on supportive medical care and weekly esophageal dilatations were carried out. At these dilatations, through the esophagoscope, it was noted that although the esophagus would be dilated to about No. 16 French, the mucosa remained very swollen, granulations were present, and there was rather profuse bleeding accompanying the treatments. Because of the apparent lack of healing and the lack of effective dilatations, we felt that the child's condition might be improved by gastrostomy, which would give a better control of her diet and would give more effective dilatations by use of the retrograde method.

In January of 1946 a gastrostomy was performed, and the child was placed on a high caloric diet. She immediately began to gain weight and showed satisfactory progress. Weekly retrograde dilatations were then commenced.

In the following year of 1947, 31 such dilatations were carried out. In 1948, 37 dilatations were done, and up to June 14, 1949, 13 dilatations were performed. The schedule of treatments was interrupted at times by intercurrent childhood diseases. In the latter years the child was ambu-

latory and came to the hospital for these treatments. The greatest dilatations ever accomplished was the passing of a No. 22 French bougie. On attempting to continue the dilatations at longer intervals the esophagus quickly closed down within two to three weeks and no food could be taken.

During the first part of 1949 definite consideration was given to surgical correction of her difficulties for the following reasons:

1. The child had by this time reached school age and naturally was very handicapped by having a gastrostomy. The psychological effect of such a condition was felt to be very detrimental to a young child at the time when she was first associating with other children.

2. There was no prospect that she would ever be able to get along without a gastrostomy and it was impossible to maintain a functional esophagus without dilatations at regular intervals of two weeks, and this would very probably be true for the rest of her life.

The hazards of the surgery involved were fully explained to her parents and were accepted by them.

On Jan. 6, 1950, this patient was submitted to surgery. The following are the surgical notes on this operation:

Under sodium pentothal, nitrous-oxide-oxygen plus ether, cyclopropane and curare anesthetic the abdomen was entered first and the stomach freed from all its attachments, its blood supply being ligated except that coming from the right gastric and the right gastroduodenal arteries. The left gastric artery was ligated close to its origin from the celiac axis. The esophagus was freed from the diaphragm and dissected blindly with the finger for a distance of about two inches up into the lower portion of the mediastinum. Following this, all bleeding points being controlled with fine silk ties, the abdominal incision was closed in layers of interrupted sutures of silk. The patient was turned on her left side and the right chest entered after removing a large segment of the right fifth rib. The thoracic esophagus was dissected from its bed throughout its entire length from the top of the chest down to the diaphragm. Bleeding points were secured with fine silk sutures. The esophagus was then resected, cutting it off at its junction with the stomach and closing the orifice into the stomach with a continuous chromic catgut suture reinforced with interrupted sutures of silk. The resected portion of the esophagus was examined and it was found that more of the esophagus had to be removed on the proximal side of the resected portion. This placed the most distal portion of the remaining proximal end of the esophagus about flush with the dome of the right chest. The stomach was then pulled up as far as possible into the right chest and secured with interrupted sutures of silk to the pleura. A circular opening was made on the anterior surface of the cardia of the stomach, and a side anastomosis was made in two layers of interrupted sutures of fine silk, 3-0 and 4-0. One layer included the mucous membranes of both stomach and esophagus, the other included the muscularis of both organs. Following this procedure, a duodenal tube was passed into the stomach. The thoracic cavity was washed out with zephiran and saline. One hundred thousand units of penicillin and 0.5 gm. of streptomycin were left in the pleural cavity. The chest wall and the various layers of muscle were closed with interrupted sutures of silk. Interrupted sutures of silk were used in the skin.

The following is the histologic report on the resected esophagus:

The surface epithelium is intact; submucosa markedly collagenous. The muscle fibres of the mucus muscle coat are distinctly hyperplastic. Most of the wall is actually formed by hypertrophic muscle fibres, and in these portions there is extensive scarring involving the inner muscle coat and the entire submucosa. There is also an older sinus tract within the thickened wall infiltrated by leucocytes and containing numerous capillaries. Extensive inflammatory fibrosis and chronic inflammatory infiltrations are also seen in the outer coat beyond the external muscle coat. In the mid and upper portions similar change with unusually marked fibrosis scarring throughout the submucosa and muscle coats are present. The uppermost portion shows much less scarring, but considerable edema.

This child made a rather uneventful convalescence considering the radical nature of the surgery and was taking fluids in three days. On Jan. 15 all sutures were removed and the thoracic and abdominal wounds were well-healed. Subsequent esophagoscopy showed good healing at the anastomosis with no strictures. This child has been followed since this time and has been able to eat quite normally.

M. S., age two years, was admitted to the Buffalo Children's Hospital on June 17, 1946, with the chief complaint of difficulty in swallowing. On June 2, 1946, this child was playing about the kitchen and found an open can of lye. He apparently placed an unknown quantity of this ma'erial in his mouth. He was taken to a hospital and given medical care for the first nine days. During the entire hospital stay the child was able to take fluids orally without difficulty. After observation he was then discharged on June 14, 1946, without any dilatations. After arriving home the patient began to have considerable difficulty in swallowing, and was then brought to the Buffalo Children's Hospital.

Physical examination revealed a well-nourished, well-developed, white male child not appearing acutely ill. He appeared quite dehydrated. The general physical examination showed no significant findings. The impression was that of esophageal stricture due to poison (lye). X-ray findings as of June 17, 1946, showed a rather ragged mucosal membrane in the middle third of the esophagus. There was one point of narrowing at the level of the fifth dorsal vertebra. The esophagus was wide both above and below this point. The length of the partially narrowed area being about 4 cm.

On June 19 the patient was taken to surgery and the esophagus was dilated to No. 17 French. There was considerable bleeding from the esophagus at the end of the procedure, indicating marked granulations. The patient left the table in good condition. Following dilatation of the esophagus at this time the patient's temperature was elevated. No apparent cause was found; however, on June 25 the patient's condition was such that he was again taken to surgery and the esophagus dilated to No. 22 French. Following this, he ate considerably better. This child was subsequently dilated every few days and at no time were we able to pass a bougie greater than a No. 22 French.

Particular trouble was noted in dilating the lowest stricture in the esophagus. After the last dilatation it was felt that the child was not passing sufficient food through the esophagus. After consul'ation a high gastrostomy was performed after the fashion of Wetzel. Postoperatively the child seemed to do very well. He was then put upon proper feeding,

given some blood, and frequent retrograde dilatations were begun. Again, at no time, were we able to dilate beyond a No. 22 French. Occasional esophagoscopies were performed, and it was noted that the esophagus remained in a state of marked inflammation with considerable easily bleeding granulations. By April 5, 1950, after many dilatations we were still unable to dilate the esophagus more than a No. 22 French. On his eighty-sixth admission to the hospital for successive dilatations it was noted that any attempt to space these dilatations at reasonable intervals was immediately attended by progressive and rapid contractures of the esophageal constrictions so that we felt that this child had nothing more than a lifetime of constant dilatations ahead of him. After consulting with the surgeons and pediatricians it was agreed that removal of the esophagus from the stomach to the point at the root of the neck with subsequent anastomosis of the stomach to the upper segment of the esophagus should be done. The parents of the boy readily consented to this operation and therefore on April 26, 1950, the following operation was performed:

Under nitrous-oxide-oxygen and ether the abdomen was entered first and the stomach was entirely freed from all of its attachments. This included a devascularization of the stomach except for the right gastric and the right gastroepiloic artery. The left gastric artery was ligated close to its base, and the stomach entirely freed from the duodenum up to the esophageal hiatus of the diaphragm. Bleeding points were all tied with fine silk and the abdominal incision was closed in layers of the same type of suture material. The right chest was then entered through the oblique incision previously mentioned, a large segment of the sixth rib being excised. The esophagus, being ligated with fine silk, and the stomach then delivered into the right chest through the esophageal hiatus. The entire thoracic esophagus, except for the upper one-half inch, was excised and an anastomosis made between the remaining proximal portion of the esophagus and the anterior wall of the stomach. This anastomosis, however, still left a very narrow stricture about one inch proximal to the level of the anastomosis. Had this been excised, it would have been necessary to make the anastomosis up in the neck. After consultation with the endoscopist at the time of the operation, it was felt that this stricture was relatively unimportant and could be easily controlled with dilatation in the future. The anastomosis was of two layers, both layers being of interrupted sutures of silk. At completion of the procedure, the right thoracic cavity was washed out with saline, catheter drainage was established and the thoracic incision closed in layers with interrupted sutures of silk. A duodenal tube was inserted through the nose into the stomach past the line of anastomosis.

The patient had a very stormy postoperative course, due mostly to obstruction at the pyloric end of the stomach. It was felt that the most likely reason for this was the effect of a vagotomy at the time of the esophagectomy, but one could not be sure that there was not some kinking or obstruction of the pylorus at the site of esophageal hiatus in the diaphragm. This condition necessitated a second operation on May 3, 1950. A right peramedian upper abdominal incision was, therefore, made and the upper portion of the abdominal cavity explored. There was no twist in the duodenum or the stomach. There was apparently no mechanical reason why the passage of gas and fluid from the stomach into the duodenum should not take place. One, therefore, had to conclude that the patient's difficulty was entirely on a functional neurogenic basis due to the division of the vagus nerves.

A Wetzel type of jejunostomy was performed for feeding in a proximal loop of the jejunum. This operation was performed through an additional incision in the left flank. The patient withstood the operation very well. It was felt at the end of the operation that we could take our time now and wait for the natural function of the stomach to return.

The child made an uneventful recovery and was discharged from the hospital on June 1, 1950. At this time he was able to take oral feeding without difficulty, and the abdominal and chest incisions were well-healed. He has been followed at intervals since the operation and he leads a perfectly normal life with no picture of esophageal trouble.

Tumors of the trachea are not common nor is there evidence of an increase incidence such as we find in bronchogenic tumors; however, enough of these difficult problem cases arise in any large medical center to cause deep concern to those responsible for their management. In 1945, Tinney *et al.*[20] reported 27 tumors of the trachea seen in the Mayo Clinic in the past 25 years. Eleven of these tumors were squamous cell carcinoma, eight were cylindromas, six were adenocarcinomas, and two hemangioendotheliomas. The location of 15 lesions were in the lower section of the trachea, eight in the upper, and one in the middle trachea. Three were too extensive for the site of origin to be determined.

The chief symptoms of cough, wheeze and dyspnea usually appear, as a general rule, quite late in the disease, and any other symptoms are too vague to receive special examination; therefore, we must expect to see tracheal tumors only in the later stage.

Treatment of tracheal tumors has at best been unsatisfactory, due to the difficulty of restoration of the airways after complete surgical removal. Small tumors of the cervical trachea have been treated by electrocoagulation through an incision into the trachea. Tumors of the lower trachea have been treated by the same method through a bronchoscope. Many of us have had the discouraging experience of removal by biting forceps, of large obstructing tumors of the lower trachea, only to see rapid recurrence with even more difficult removal until finally such procedures become impossible, and death by suffocation was the end-result. We have also had, what I might call the pleasure of removing adenomas and benign lesions by this method and have prolonged life for years;

however, even in some of these cases, deep extension with resulting constriction of the airway proved too much for our efforts. Just recently we have seen a case of adenoma arising in the trachea, near the carina, which was held in check for several years, by piecemeal removal through a bronchoscope; but which was at last causing obstruction to both main bronchi by extension beyond the carina.

It is, therefore, with much satisfaction that the endoscopists note the encouraging experiments and clinical work being done by those surgeons interested in these problems.

Successful resection of small portions of the trachea have been done for some years. Ferguson notes that several such operations involving removal of small segments of the trachea were reported in the German literature from 1896 to 1909. When possible, only portions of the trachea have been removed, and the defects corrected by various methods, such as skin flaps, stiffened by bone, cartilage or metal wires, silver gauze, tantalum mesh or fascia. All of these have been used with some success. Rigid tubes of glass, stainless steel and vitalum have also been used for bridging defects. Clagget *et al.*[21] have recently used polythene tubes in their study of tracheal resections in dogs. This material seemed especially desirable because water has no effect on it and because blood and mucus do not adhere or clot quickly in the tube. They were able to remove segments of the trachea averaging five rings and successfully bridge the gap with this material. They also reported a tracheal resection in a patient which included the carina and one lung with apparent technical success; however, death occurred on the sixth day due to pneumonia. Partial blocking of the left upper lobe bronchus by the polythene tube was probably the initiating factor.

Ferguson *et al.*[22] have carried out extensive animal experiments, performing 58 operations on dogs. They developed a technique affording accurate approximation of the mucosa and found no severe strictures resulting after sections of the trachea had been removed. They also developed a method of working with a tracheobronchial anesthetic tube extending into one bronchus, so that carinal resection with pneumonec-

tomy could be done quite successfully; however, all these attempts of reconstruction of the tracheal segments using tracheal monografts, autografts, aortagrafts and tantalum gauze were unsuccessful. Ferguson concludes that, "The comparative ease and safety of tracheal resection and anastomosis suggests that where indicated. tracheal resection may properly be used in place of less effective measures."

Dr. Macmanus recently published a report on a case of tracheal resection as follows:

L. B., age 61, white male, first seen on Jan. 16, 1952. This man's chief complaint was occasional hemoptysis during upper respiratory infections. X-rays and physical examination revealed no significant pathology. Bronchoscopy, however, disclosed a mass on the anterior tracheal wall about 2 cm. above the carina. Its apparent base measured approximately 2.5 cm. The mass was irregular, soft, did not bleed easily and did not significantly occlude the trachea. A biopsy was taken and diganosed as a primary carcinoma of the trachea. Because of the nature of this tumor and its location, resection of the trachea was advised, and the operation was accepted by the patient. The nature of the operation was as follows:

A long incision was made, parascapular in type, and the fifth rib resected. The chest was opened widely and the lung packed downward and the esophagus exposed through the mediastinal pleura. A careful dissection of the posterior mediastinal lymph nodes was made. None of these lymph nodes seemed to be clinically involved with the tumor. The trachea was freed from the esophagus and hemostasis was carefuly secured. The trachea was then opened just below the tumor, and an intratracheal tube was directed into the left main bronchus and its cuff inflated, thus giving very good respiratory exchange in the left lung. Under direct visualization a sleeve resection of the trachea was performed, removing three rings anteriorly, the section removed measuring about 3.5 cm. in length. Due to the extensive mobilization of the trachea, it was felt that the severed ends might be brought together without undue tension. The tracheal walls were reunited by interrupted sutures of 3-0 silk. In order to protect the anastomosis a section of fascia-lata which had already been prepared, was wrapped around the anastomosis and snugly sutured about the trachea from the bifurcation up to the thoracic inlet. The mediastinum was carefully closed and extrapleural drainage provided for the mediastinum with the use of a rubber catheter.

The patient's postoperative course was entirely uneventful, and the tracheotomy tube which had been put in place following the operation was removed on the seventeenth day. A bronchoscopy was performed on that same day and this showed almost complete epithelialization at the site of the anastomosis. He was discharged one month after surgery and has remained asymptomatic. Further studies of the tumor revealed it to be a cylindroma. Dr. Macmanus concluded from this case and others that approximately one-third of the tracheas might be resected. He also stressed the important factors in this type of operation:

"*1.* Adequate mobilization of the trachea up to the apex of the chest and well down over the carina.

"*2.* Adequate control of the anesthesia by directing either a separate tube through the chest into the carina and dependent lung or using the intratracheal tube as a closed circuit by delivering it

directly into the dependent main bronchus in order to have adequate control of the respiration of at least one lung during the procedure.

"*3.* Sharp, accurate section of the segment of the trachea to be removed so that when approximated the cartilaginous portion of the semirigid trachea will fit snugly and give natural apposition to the proximal and distal segments and thereby prevent troublesome air leaks.

"*4.* Support of the suture line with a large fascial graft sleeve to insure its stability and integrity.

"*5.* The use of a complementary tracheotomy to reduce the tidal air, to protect the suture line in the trachea from excessive pressure during coughing, and to allow easier aspiration of the lower trachea and upper bronchi during the early postoperative period."

We have recently had under our observation a young lady with an obstructing adenoma of the trachea arising just above the carina. For more than two years this patient had been kept free from obstruction by piecemeal removal of parts of the tumor through a bronchoscope. Nevertheless, she had recurrent attacks of pneumonia, and at examination we found this tumor to have extended down beyond the carina, involving the upper portion of both the right and left bronchi. The patient was advised to have surgical removal and she readily consented. She was taken to surgery and Dr. Macmanus removed the lower portion of the trachea and the entire carina along with the upper portions of both main bronchi. He successfully formed a new carina by anastomosing the two approximal portions of the main bronchi; however, the extent of the lesion had necessitated removal of so much of the trachea that he could not secure a direct anastomosis of the trachea and the bronchi. He, therefore, used the Belsey type of plastic repair consisting of a wide strip of fascia-lata reinforced by thin steel wire. Unfortunately, a small leak occurred in the right lateral portion of the reconstructed tracheal repair and subsequently empyema and multiple abscesses occurred, causing the death of the patient. A postmortem examination showed that the newly constructed carina had healed by first intention and that the only point of failure was the leak occurring at the tracheal end of the reconstruction where the fascia-lata was used.

I am using these two cases to point out that those occasional tracheal tumors we have all seen and which have appeared hopeless may now be given some hope for life if a tracheal resection is called for before the lesion becomes too extensive.

In this paper, I have endeavored to point out the three types of cases that are commonly seen and treated by endoscopists. In the vast majority we have had excellent results; however, there is a small percentage, in which our efforts have not been equal to producing a cure, and where the newer methods of thoracic surgeons with their increasing competence, have given great benefits by surgery. There are probably also borderline cases where over a long period of time we can get fairly good results, but where the effects would be much greater and quicker if surgical methods were instituted.

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TERRAMYCIN AND SURGERY IN A CASE OF OSTEOMYELITIS OF THE FRONTAL SINUS. CASE REPORT.

MORRIS HYMAN, M.D.,
Cincinnati, Ohio.

Osteomyelitis of the skull may be considered as one of the treacherous complications of frontal sinusitis. Young persons whose cranial bones show large diploe are more subject to osteomyelitis than older patients. Infection may extend by diploetic erosion, particularly in the spongy layer close to the mucosa and also by thrombophlebitis, that is, by isolated foci.

Surgery is indicated as soon as symptoms and Roentgenographic findings show bone destruction. Antibiotics offer a supporting therapy which is invaluable; yet, the osteolytic process may be quiescent during therapy and later may flare up upon withdrawal of the antibiotic. It is imperative that such treatment be maintained until complete cure is accomplished. This is demonstrated in the following case:

J. D., white man, age 29, machinist, was seen on March 28, 1951, complaining of severe frontal headaches. A relevant history included sinusitis in 1942. At that time, a left antral lavage yielded thick yellow pus. Nasal polyps were removed about that time. A tonsillectomy was performed in May, 1942.

He had occasional attacks of sinusitis while in the army during World War II.

Examination showed small polyps in the left anterior middle turbinal area; no pus was observed. The polyps were removed. Intermittent pain in the frontal areas did not interfere with sleep. On April 23, 1951, he showed slight edema of the upper left eyelid and he complained of nocturnal pain. Pressure on the floor of the left frontal sinus elicited moderate pain. Terramycin (500 mg. every six hours) was started on April 27. Tenderness and pain was relieved to tolerance, but the edema of the lid persisted. This patient had continued to work and refused hospitalization; however, on April 30, 1951, he consented to go to the hospital. Roentgenograms on May 1 showed osteomyelitis of the left frontal sinus (anterior plate). Under I.V. pentothal anesthesia an incision was made under the left eyebrow. About 2 drams of thick yellow pus were removed.

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(Culture was negative.) A curette fell into the frontal sinus cavity. The anterior wall was soft and necrotic for an area about the size of a quarter. This was carefully curetted until firm bone was encountered. The nasofrontal duct was widened; the wound was packed.

The patient became comfortable and free from pain. The pack was removed three days after the operation. The patient was discharged on May 7 (six days postoperative) and the terramycin was discontinued.

On May 11 edema of the lid recurred. The wound was separated by a hemostat and a wick introduced. Terramycin was started again (250 mg. every six hours) and continued until June 10 (40 days postoperative). At that date the patient was discharged as cured.

A virus type of infection associated with a rhinitis occurred in November, 1951, but without sinus complications. The patient has remained well to date.

In conclusion, this case demonstrates the necessity for extended antibiotic therapy in conjunction with and as an adjunct to surgery of the frontal sinus.

BOOK REVIEW.

Hearing Aids. Their Use, Care and Repair. By Matthew Mandl, Lecturer in Electronics and Television, Technical Institute, Temple University. 158 pages with Index. New York 11: The Macmillan Co., 60 Fifth Avenue. 1953. Price $3.50.

This book has been read in whole or in part by several members of the staffs of the Hearing Clinic and the Research Department of Central Institute for the Deaf. These readers have commented on it to the present reviewer from the point of view of their special interests and competencies. The book fared better at the hands of the physicists and engineers than with the audiologists and clinicians, which is not surprising as the author is himself a Lecturer in Electronics and Television. He seems to be writing for the hearing aid dealer, or for teachers and parents of children who wear hearing aids and give them hard use. His technical expositions are clear and well conceived.

The author steps out of his field, however, in his discussion of types of deafness and other clinical matters. Actually the book would have been better without Chapters 2, 3, 4 and 10. It would then have lived up to its title but would still have stayed within the author's area of authority and competence. The following excerpt represents the low point of the book and (together with the over-enthusiastic jacket blurb) constituted the target for the most vigorous criticism among the reviewer's associates:

> ". . . Otosclerosis may consist of scar tissue developed because of former punctures in the eardrum as well as tissue growth between the middle ear and the inner ear which limits the free vibration of the eardrum or other organs of the inner ear.
>
> "When an audiometer is utilized with an air conduction receiver, the loss indicated is of the conductive type. This is particularly true if the loss is greater than approximately 30 db. . . ."

Such careless writing (or thinking?) undermines a reader's confidence in other parts of the book. The doubt is likely to be confirmed by the last sentence of the Appendix on the decibel, which reads —

> "In audiometer work, the reference level of zero is based on the sound intensity just perceptible by the average person with normal hearing."

No further explanation is given of the concept of hearing loss or the audiogram, and no explanation is given of the zero reference level in the table immediately following in which the threshold of average hearing is given at 10 db, not zero. The electrical reference level of 6 millivolts is explained but not the acoustic reference level of 0.0002 microbar. The selection of this electrical reference level as the one to talk about shows clearly the author's orientation to electrical engineering rather than to acoustics or audiology.

The reviewer was surprised to find particular makes of hearing aids and batteries, etc., mentioned by name in the text and in the legends of figures and hopes that the manufacturers whose names were omitted will not be too severely disappointed.

All in all, the practical chapters on the description, care and repair of hearing aids are good, but the prospective reader should not be misled by the jacket blurb into thinking that he will find the answer to the question, printed in bold-faced type, "What type of hearing aid will be most effective for me?" H. D.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSE.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, Executive Secretary-Treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

XVIII CONGRESSO INTERNAZIONALE OTO-NEURO-OFTALMOLOGICO.

The XVIII International Congress of Oto-Neuro-Ophthalmology will be held in Bologna, Italy, May 3-7, 1953. Chairman: Prof. Q. Di Marzio. Official reports are:

1. Dysrraphia in Oto-Neuro-Ophthalmology: General Reporter: Belloni, Padua. Embryology: Krabbe, Köbenhavn. Neurology: Vercelli, Milan. Radio-Neurology: Mascherpa, Milan. Oto-Laryngology: Arslan, Padua. Ophthalmology: Franceschetti, Geneva. Neuro-Surgery: Fasiani, Milan. Facial-Surgery: Sanvenero-Rosselli, Milan.

2. Ménière's Disease: Reporters—Neurology: Barré, Strasbourg. Otology: Greiner, Strasbourg. Neuro-Surgery: Tolosa, Barcelona.

The official languages admitted are: Italian, English, German, French, Spanish.

Besides the above mentioned subjects, papers will be also accepted on arguments regarding at least two of the specialties at the same time.

For further information address Dr. Giuseppe Cristini, Clinica Oculistica-Policlinico, Bologna, Italy.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

April 1, 1953.

Audicon Models 400, 415, 530 and 615.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Roosevelt Hotel, New Orleans, La., Apr. 20-24, 1953.
Palmer House, Chicago, Ill., Oct. 5-9, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

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Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 28-29, 1953 (afternoons only).

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Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

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Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater, Miss., May 11, 1953.

OTOSCLEROSIS STUDY GROUP.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
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Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

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Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Meeting:

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President: Dr. D. A. Morgan, Norfolk, Va.
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Spring Meeting: Hot Springs, Va., May, 1953.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Orwyn H. Ellis, M.D.
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Secretary of Section on Ophthalmology: Sol Rome, M.D.
Chairman of Section on Otolaryngology: Leland R. House, M.D.
Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

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Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21, N. Y.

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Secretary and Trasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Charleston, S. Car., Sept. 13-16, 1953.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clay W. Evatt, Charleston, S. Car.
Vice-President: Dr. David S. Asbill, Columbia, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.
Meeting: Los Angeles, Calif., 1953.

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Meeting:

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Meeting: Hot Springs, Va., May 2-3, 1953.

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CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ontario.
Time: June 14-18, 1953.

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President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: Second International Congress of Bronchoesophagology.
Time and Place: June 15 and 16, 1953, Albert Plage, Le Zoute, Belgium.

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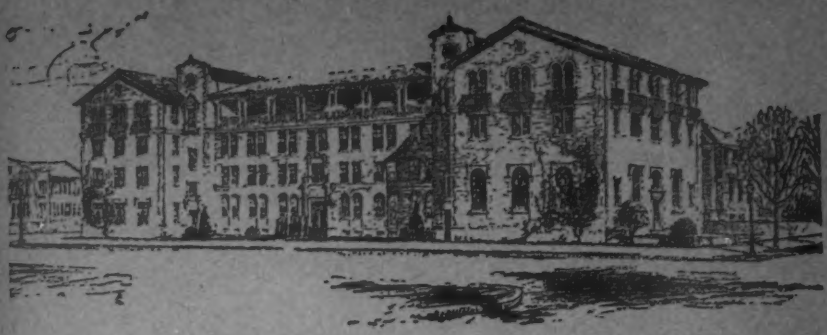

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